**Conclusion:** New-onset gout was independently associated with a 51% increased risk of return to dialysis >12 months after primary KT compared to a control cohort without gout. To our knowledge, this is the first observation of this outcome in an appropriately controlled cohort study of KT recipients with gout. Results from this analysis may have important implications for the monitoring and management of new-onset gout in the kidney transplant population.

**References:**

[1] Mandell BF. *Cleve Clin J Med* 2008;75(Suppl 5):S5-8.
[2] Forbess LJ, Fields TR. *Sem Arthritis Rheum* 2012;42:146-54.
[3] Gibson T. *Curr Opin Rheumatol* 2012;24:127-31.
[4] Zhang L, et al. *Nephrol Dial Transplant* 2012;27:1836-9.
[5] Clive DM. *J Am Soc Nephrol* 2000;11:974-9.
[6] Kalantar E, et al. *Transplant Proc* 2011;43:584-5.
[7] Lin HY, et al. *N Engl J Med* 1989;321:287-92.
[8] Ben Hmida M, et al. *Transplant Proc* 1995;27:2722-4.
[9] Kanbay M, et al. *Transplant Proc* 2005;37:3119-20.
[10] Baroletti S, Bencivenga GA. *Prog Transplant* 2004;14:143-7.
[11] Kim ED, et al. *Am J Transplant* 2015;15:482-8.
[12] Kim DG, et al. *PloS One* 2018;13:e0209156.

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## THU0409 A RANDOMIZED, PHASE 2 STUDY EVALUATING THE EFFICACY AND SAFETY OF ANAKINRA IN DIFFICULT-TO-TREAT ACUTE GOUTY ARTHRITIS: THE ANAGO STUDY

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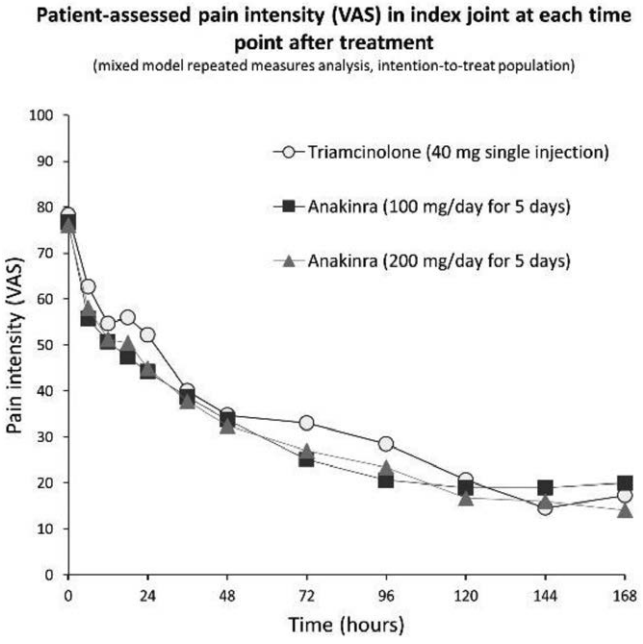
**Background:** In gout, urate crystals deposited in and around joints trigger episodes of acute arthritis, mediated by the proinflammatory cytokine IL-1β. In uncontrolled studies, the IL-1 receptor antagonist anakinra appears effective in reducing pain and signs of acute flares in patients with difficult-to-treat gout. However, confirmatory, adequately-powered, prospective trials are lacking. The 'anaGO-study' (anakinra in gout) was a multi-center, randomized, double-blind, double-dummy, phase 2 study investigating the efficacy and safety of anakinra in acute gout (NCT03002974).

**Objectives:** The primary objective was to evaluate the efficacy of two regimens of anakinra (100 or 200 mg daily s.c. injections for 5 days) compared to triamcinolone (single i.m. injection 40 mg) with respect to patient-assessed pain intensity. The primary endpoint was change in pain intensity from baseline to 24-72 hours (average of 24, 48 and 72 hours) in the most affected joint measured on a visual analogue scale (0-100 VAS). Secondary outcomes included: time to onset of effect, time to response, time to pain resolution, time to rescue medication use, patient's and physician's assessments of global response, clinical signs, inflammatory biomarkers and safety.

**Methods:** Patients were recruited who had acute gout based on ACR/EULAR 2015 gout classification criteria, and were unsuitable for anti-inflammatory therapy with NSAIDs and colchicine due to contraindication, intolerance or inefficacy. Patients were randomized to each group in a 1:1:1 ratio and stratified by urate-lowering therapy use (yes/no) and BMI (<30.0 or ≥30.0 kg/m$^2$).

**Results:** 165 patients were randomized; 110 to anakinra (56 to 100 mg/day, 54 to 200 mg/day) and 55 to triamcinolone; 108 and 53 were included in the primary analysis, respectively. The median (range) age was 55 (25-83) years, 87% were male, mean disease duration was 8.7 years and mean number of self-reported flares during the past year was 4.5. The pain intensity, from baseline to 24-72 hours, decreased in both treatment groups; mean (95% CI) change was -39.4 (-46.8, -32.0) for triamcinolone and -41.2 (-46.3, -36.2) for anakinra. The 100 mg and 200 mg doses of anakinra were comparably effective in decreasing pain (100 mg/day: -41.8 [-48.9, -34.8] and 200 mg/day: -40.7 [-47.9, -33.4]). Mean (95% CI) difference in pain reduction between anakinra and triamcinolone treatment groups was -1.8 (-10.8, 7.1) (p-value = 0.688 for primary endpoint). The majority of secondary efficacy endpoints were numerically in favor of anakinra, and in most instances also statistically significant, in comparison to triamcinolone, e.g. physician's assessment of clinical signs at 72 hours and patient's and physician's assessment of global response at Day 8. No unexpected safety findings were identified in any of the treatment groups.

**Conclusion:** Anakinra and triamcinolone reduced patient-assessed gout flare pain to similar degrees in patients for whom conventional therapy was ineffective or contraindicated. Both doses of anakinra showed comparable efficacy in pain reduction. The majority of secondary efficacy endpoints favored anakinra. Anakinra was shown to be an additional option for use during acute gout flares.

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## THU0410 COMPANION IMMUNOSUPPRESSION WITH AZATHIOPRINE INCREASES THE FREQUENCY OF PERSISTENT RESPONSIVENESS TO PEGLOTICASE IN PATIENTS WITH CHRONIC REFRACTORY GOUT

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**Background:** Pegloticase is a mammalian recombinant uricase coupled to monomethoxy polyethylene glycol that is approved in the US for treatment of patients with chronic refractory gout and causes profound reductions in serum urate. However, treatment with pegloticase is limited by the induction of anti-drug antibodies and loss of responsiveness in nearly half of treated patients.

**Objectives:** The goal of this study was to determine whether co-therapy with azathioprine (AZA) would increase the frequency of chronic refractory gout patients who had persistent urate lowering from pegloticase therapy.

**Methods:** This open label multicenter study enrolled subjects with chronic gout who failed to lower serum urate to <6 mg/dL despite medically indicated doses of urate lowering therapy (NCT02598596). Patients were screened for adequate levels of the AZA metabolizing enzyme thiopurine methyl transferase and then started on daily oral AZA 1.25 mg/kg for 1 week and then 2.5 mg/kg for